

Diagnosis and management of blunt cerebrovascular injuries: What you need to know

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ABSTRACT: Blunt cerebrovascular injuries are not as rare as they were once thought to be—but they still have the same potential for disastrous outcomes. They may occur following any trauma, but more common with higher energy transfer mechanisms. If stroke occurs, prompt recognition and treatment offers the best chance for optimal outcome. Early diagnosis and provision of antithrombotic therapy may prevent strokes, so screening of asymptomatic patients is recommended. Herein we will present what you need to know to diagnose and manage blunt cerebrovascular injury. (*J Trauma Acute Care Surg.* 2025;98: 1–10. Copyright © 2024 Wolters Kluwer Health, Inc. All rights reserved.)

KEY WORDS: Blunt cerebrovascular injury; blunt carotid artery injury; blunt vertebral artery injury; endovascular; stroke.

HISTORICAL PERSPECTIVE

Thirty years ago, blunt carotid artery injuries (BCI) and blunt vertebral artery injuries (BVI)—collectively referred to as blunt cerebrovascular injuries (BCVI)—were considered rare and devastating.^{1,2} Multicenter studies reported a BCI incidence of 0.1% among blunt trauma admissions.^{3,4} Nearly all patients were symptomatic at diagnosis and the mortality was 23% to 28%, with 48% to 58% of survivors suffering permanent severe neurologic deficits. Screening of asymptomatic patients began in the mid-1990s,⁵ and the reported incidence and outcomes have markedly changed. Injuries are currently diagnosed in 1% to 3% of blunt trauma admissions; the large majority are asymptomatic, and stroke rates and neurologic outcomes have improved considerably.

ANATOMIC CONSIDERATIONS

Anatomic variations are common in the cerebral circulation.^{6,7} The anterior and posterior circulation connect through the Circle of Willis, which is intact and symmetric in only 20% of people.⁷ The carotid arteries and their branches comprise the anterior circulation, which supplies the majority of the cerebral hemispheres. Typically, the right common carotid artery (CCA) originates from the innominate artery and the left CCA from the aortic arch. At the superior border of the thyroid cartilage, the CCA divides into the internal (ICA) and external carotid artery (ECA).

The ECA has multiple branches that supply the face, scalp, and collateral cerebral circulation. The ICA is divided into four segments: cervical, petrous, cavernous, and cerebral. The cervical ICA typically has no named branches as it ascends anterior to the spine. The petrous segment passes through the carotid canal in the petrous portion of the temporal bone. The cavernous segment runs through the cavernous sinus within the cranial vault; because of its S-shape it is referred to as the carotid siphon. The cerebral segment begins where the ICA passes through the dura mater and divides into the anterior (ACA) and middle (MCA) cerebral arteries.

The vertebral arteries (VAs) and their branches comprise the posterior circulation, supplying the brainstem, cerebellum, and posterior cerebral hemispheres. The VAs usually originate from the subclavian arteries and are divided into four segments. The V1 segment courses anterior to the scalene muscle to enter the transverse foramen of the C6 vertebral body. The V2 segment traverses the transverse foramina from C6 to C2. The V3 segment runs from the C2 transverse foramen to the skull base. Intracranially, the V4 segment joins the contralateral VA, forming the basilar artery. The VAs are commonly asymmetric, with hypoplasia (defined as diameter <2 mm and asymmetry ratio <1:1.7) in 16% of patients, usually (66%) on the right.⁸

PATHOPHYSIOLOGY

Blunt cerebrovascular injuries result from forces that stretch, twist, or otherwise impact the artery, causing tearing of one or more layers of the arterial wall or an intramural hematoma. In 1974, four mechanisms of BCI were described.⁹ A “direct blow” (e.g., hanging, strangulation, dog bite) was designated a Type I injury. Type II injuries, the most common, are due to cervical hyperextension, flexion, or rotation. In hyperextension or rotation the ICA may be stretched over the lateral articular processes of the C1–C3 vertebral bodies. In flexion the artery may be compressed between the mandible and the vertebral bodies.¹⁰ Type III injuries involve intraoral trauma, such as a child falling with a toothbrush in the mouth (Supplemental Digital Content Figure, <http://links.lww.com/TA/D953>). Type IV injuries are caused by a basilar skull fracture, usually in the carotid canal.

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The relationship between BCVI and a high-energy transfer mechanism is well-known, and the risk appears to increase with increasing energy transfer.^{3–5,11} However, the specific threshold of energy is unknown and a wide variety of trauma mechanisms—some seemingly minor—have been associated with BCVI. For example, in patients older than 65 years, 67% of BCVI result from ground-level falls.¹² There is a body of literature on BCVI following “trivial trauma” such as chiropractic manipulation; visiting the hairdresser; bottoms-up drinking; head-banging (“rock-n-roll neck”); and virtually every sporting activity.⁶ The trivial trauma literature intersects with that of “spontaneous dissections.” Risk factors for spontaneous dissections include hypertension, fibromuscular dysplasia, syphilis, Marfan syndrome, Erdheim’s cystic medial necrosis, Ehler-Danlos syndrome type IV, and other arteriopathies. These risk factors may predispose patients to BCVI following low-energy trauma.⁶

Although commonly referred to as “dissections,” BCVI present in a variety of forms (Fig. 1). Intimal injury exposes thrombogenic subendothelial collagen, triggering platelet aggregation and thrombus formation. The resultant clot may occlude the vessel or embolize distally. Intramural hematoma, dissection, or pseudoaneurysm may progress to completely occlude the lumen. Full-thickness arterial laceration results in active hemorrhage or an arteriovenous fistula.

CLINICAL PRESENTATION

Clinical manifestations of BCVI depend on the affected vessel, injury grade, preexisting cerebrovascular disease, and collateral circulation. Associated injuries and intoxication may compromise the detection of symptoms. Signs and symptoms of BCVI include arterial hemorrhage from neck, mouth, nose, or ears; expanding cervical hematoma; cervical bruit; new focal neurologic deficit (hemiparesis, transient ischemic attack, vertebrobasilar symptoms); Horner’s Syndrome (oculosympathetic paresis); evidence of stroke on computed tomography (CT) or MRI; or neurologic deficit inconsistent with imaging. The signs vary in their sensitivity and specificity for BCVI and may be attributed to preexisting atherosclerotic disease or other injuries, e.g., spinal cord (SCI) or traumatic brain injury (TBI). Cervical arterial dissection may be associated with neck, ear, face, or periorbital pain, but pain may be attributed to other injuries or difficult to elicit. Arterial occlusion or thromboembolization will result in deficits based on the affected territory. The manifestations of BVI may be subtle, such as ataxia and visual changes.⁶ Unilateral VA occlusion without thromboembolization may not result in any deficits if the contralateral VA is patent.

Progression of the injury and clinical manifestations are a dynamic process. Accumulation of thrombus or luminal stenosis can result in hypoperfusion, occlusion, or thromboembolization. A latent period between the time of injury and the appearance of symptoms is well described. In a study of strokes after BCVI, only 37% were evident at the time of presentation.¹³ Among those who subsequently developed neurologic deficits, 61% occurred within 72 hours (Fig. 2).

SCREENING

In the early 1990s, it was suggested that BCVI were under-diagnosed and often masked by other injuries such as severe

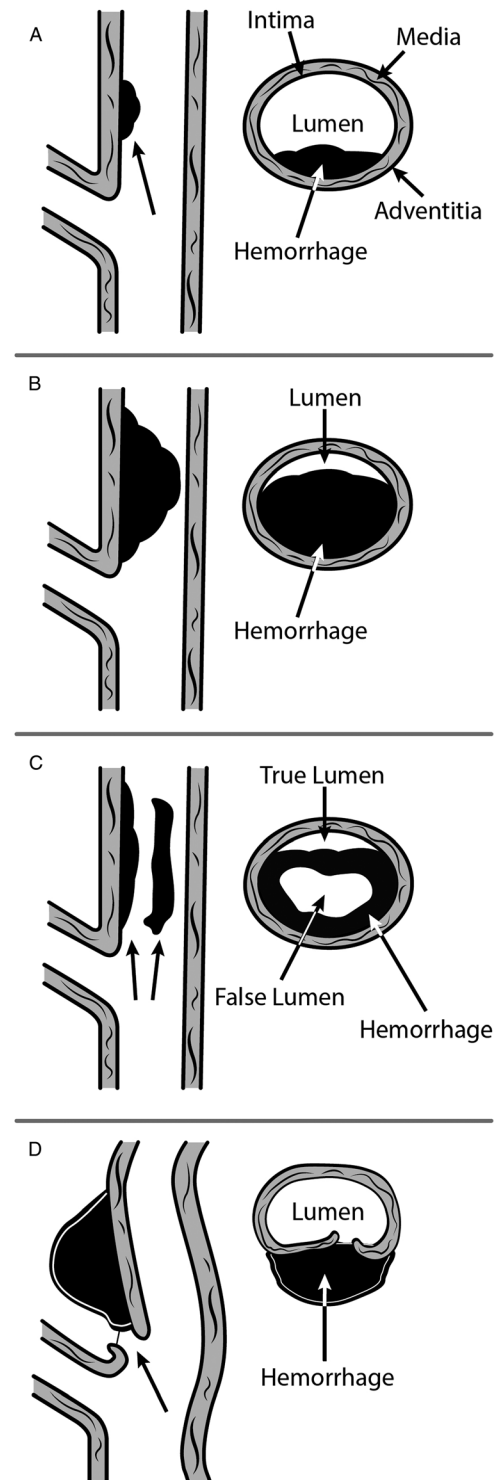


Figure 1. Forms of BCVI. (A) A small intimal tear or intramural hematoma can appear on imaging as a simple irregularity of the vessel. (B) Intramural hematoma or intraluminal thrombus may expand to narrow the arterial lumen. (C) A larger intimal tear may be associated with a dissection as the intimal layer separates from the underlying media. The dissection may narrow the lumen to the point of complete occlusion. (D) If the arterial wall is weakened a pseudoaneurysm may form, which can enlarge and compress the lumen, or potentially rupture.

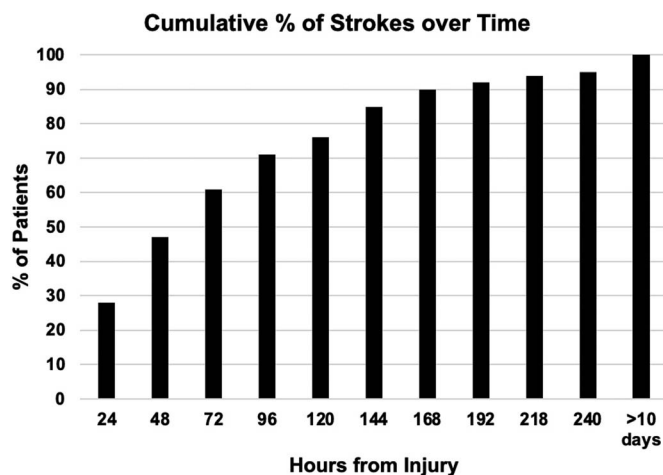


Figure 2. Cumulative time to stroke as a percentage of 144 patients diagnosed with stroke on the basis of new-onset neurologic symptoms, grouped in 24-hour intervals following presentation. Data from Burlew et al.¹³

TBI.^{3,4,11} To test this hypothesis, patients who were undergoing aortography to rule out blunt thoracic aortic injury had carotid arteriography.¹⁴ Carotid injuries were found in 6 (3.5%) of 171 patients, half of which were clinically unsuspected. The Denver group began comprehensive screening for BCI in 1996.⁵ Among 25 patients diagnosed in 17 months (incidence 0.86%), only seven were symptomatic. Five comatose patients were found to have BCI, along with 13 asymptomatic patients. By 2002, the BCVI incidence was 1.6% among blunt trauma admissions.¹⁵ Multiple centers reported similar incidences, and screening became widespread.¹⁶ The issues were patient selection, optimal imaging modality, and management of the injuries.

Screening Criteria

The original screening criteria were based on the understanding of mechanistic risk factors.^{5,9,10} They included signs and symptoms; mechanism compatible with severe hyperextension or flexion and rotation of the neck; significant anterior cervical soft-tissue injury; cervical (c-) spine fracture; displaced midface or mandibular fracture; and basilar skull fracture involving the sphenoid, mastoid, petrous, or foramen lacerum. A clinical dilemma rapidly emerged: it was impractical to liberally perform digital subtraction arteriography (DSA) or to transfer patients to trauma centers just for this purpose. Based on multiple logistic regression analysis, high-risk criteria were identified: Glasgow Coma Scale (GCS) score <6; petrous bone fracture; diffuse axonal injury; Le Fort II or III fracture; and c-spine fracture.¹⁷ The 2010 Eastern Association for the Surgery of Trauma (EAST) Practice Management Guidelines recommended screening based on high-risk criteria.¹⁸ Many studies have sought to further refine screening criteria. Some areas deserve specific comment.

Cervical Spine Injuries

Fractures/distraction injuries at all cervical levels are an independent risk factor for BVI,¹⁹ and were included in the 2009 Western Trauma Association (WTA) Critical Decisions algorithm.²⁰ The 2010 EAST guidelines¹⁸ included only high-risk patterns (C1-C3 and foramen transversarium fractures; subluxation/

dislocation injuries); however, the 2020 EAST guidelines²¹ recommended screening for low-risk c-spine injuries as well.

Traumatic Brain Injury

Patients with severe blunt TBI are presumed to have sustained cervical hyperextension, flexion, or rotation; GCS ≤6 was identified as a high-risk predictor.¹⁷ The EAST 2010 guidelines recommended screening for GCS ≤8.¹⁸ Most guidelines include neurologic abnormality incongruous with CT findings. While altered mental status associated with mild TBI may technically meet this criterion, there is no known association between mild TBI and BCVI and we do not suggest that every concussion patient be screened. The recommendation for screening for “TBI with thoracic injuries” found in the “Expanded Denver Criteria”²² is based on studies in severe TBI patients.

Facial Fractures

A systematic review identified Le Fort and mandibular fractures as the highest-risk facial fractures.²³ A recent study of 1440 patients with craniomaxillofacial fractures found a significant association with Le Fort III and pure orbital wall fractures; orbito-zygomatic ($p = 0.063$) and mandible ($p = 0.066$) fractures were close to significance.²⁴ Speaking to the importance of energy transfer, BCVI were very uncommon after assaults but much more likely in the setting of traffic accidents.

Neck Soft Tissue Injury

The incidence of BCVI in near-hanging is 3% to 6%, and the likelihood of significant aerodigestive or spinal injuries is high enough to warrant CT imaging.²⁵ Strangulation and seatbelt abrasions are less commonly associated with BCVI, particularly in the absence of symptoms or high-risk injuries, and some have questioned the need for screening.²⁶

Thoracic Trauma

An association between thoracic injuries and BCVI has been found in some multivariate analyses but not others. The expanded Denver criteria include thoracic vascular injury, cardiac rupture, and significant thoracic injury (AIS ≥3) combined with significant TBI.²²

High-Energy Transfer Mechanism

Several guidelines—including that of the American College of Surgeons (ACS) Trauma Quality Improvement Program (TQIP)—recommend screening based on a “high risk or high-energy mechanism.”^{20,22,27} It has proven extremely difficult to quantify a high-energy mechanism, and the decision to screen is often based on the clinician’s inference and gestalt.

Universal Screening

A recurring theme in the literature is that more liberal screening finds more BCVI. Many injury patterns and mechanisms pose varying degrees of risk—and none of them are zero. Limiting screening to high-risk criteria will miss at least 20% of BCVI.^{17,28} A model based on injury patterns would require screening 96% of patients in the National Trauma Data Bank in order to identify all BCVI.²⁹

Leichtle et al.³⁰ studied all major trauma activation patients, finding injuries in 2.7%. Of those, 23% did not meet expanded Denver or TQIP screening criteria—and all of them were in motor vehicle crashes (MVCs). In the most “universal” protocol to date, all patients except those with isolated extremity injuries were screened.³¹ Of note, 64% of the patients were in MVCs. There were BCVI in 7.6%, of whom 25% did not meet screening criteria.

While implementation of universal screening is likely to identify more injuries, the additional costs and potential harms need to be considered. Our current approach is to screen patients with either a high-risk injury or a high-risk mechanism (e.g., MVC >30 mph, fall >10 ft, motorcycle crash or bicycle ejection). Elderly patients who sustain ground-level falls are vexing—and very common; we tend to focus screening on those with craniofacial injuries, but it is not uniform. We recommend that centers establish screening criteria based on published guidance, and apply them in a consistent and timely manner.

IMAGING

Digital Subtraction Arteriography

Arteriography is still considered the criterion standard for imaging the cervical and intracerebral arteries. However, it is resource-intensive, not uniformly available in non-trauma centers, and associated with complications including stroke.^{15,32} Consequently, noninvasive alternatives are favored for screening.

Duplex Ultrasonography

Duplex ultrasonography was first studied in the early 1990s for BCI.⁴ It provides visualization as well as indirect evidence of injuries (obstructive waveforms, altered velocities) but such findings are limited to significant degrees of stenosis so Grade I and many Grade II injuries could be missed. Limitations include imaging at the base of the skull and visualization of the VAs. Patients with cervical soft tissue injuries may not tolerate the examination, and it requires removal of the cervical collar. The sensitivity was only 39% in one large study,³³ and it is not recommended for screening. There may be a role in evaluating select cases such as those with a direct blow to the anterior neck; for the follow-up of proximal carotid injuries; and possibly in children in whom other modalities may require heavy sedation. The test is highly dependent on the ultrasonographer and the clinician must recognize its limitations.

Magnetic Resonance Arteriography

Magnetic resonance angiography (MRA) would appear to be an excellent alternative, with no bony interference, early detection of cerebral infarction, and avoidance of iodinated contrast. Early studies and a more recent meta-analysis reported poor sensitivity and specificity and there are logistical impracticalities so it is not recommended for routine screening.^{16,34,35} High-resolution vessel wall imaging and sequences such as double inversion recovery black blood imaging may be of value in identifying high risk for stroke; differentiating anatomic variants from clinically significant abnormalities, or acute from chronic intramural hematoma.^{36,37}

Computed Tomographic Angiography

The CT scan is ubiquitous in the evaluation of trauma patients. Although early studies of computed tomographic angiography (CTA) reported poor sensitivity and specificity,^{16,34} accuracy has improved significantly as technology has advanced and it is now the test of choice for integration into the trauma workflow, accuracy, and cost-effectiveness in screening.^{32,38} Multidetector-row CT with 64 or more channels is recommended.³⁹ In the setting of an equivocal finding, we perform DSA, MRA, or repeat CTA in 48 hours to 72 hours.

INJURY GRADING

A grading scale for BCI was developed in 1999.⁴⁰ Originally based on DSA, it has been validated for CTA⁴¹ and applies to BVI.¹⁹ The distribution of BCVI (BCI vs. BVI) and grades varies based on the injured artery, energy transfer and screening criteria (Table 1). Among patients screened based on high-energy mechanisms and high-risk criteria the majority of injuries were BCI¹⁵; the universal screening population was more evenly split.³¹ By comparison, in a recent study from a center with falls as the primary mechanism of injury, BVI accounted for 70% of BCVI.⁴² The distribution of injury grades is relatively consistent for each artery, with fewer Grade III and more Grade IV injuries in BVI than BCI.^{15,31,42,43}

Stroke rates associated with BCI increase with increasing injury grade (Supplemental Digital Content Table, <http://links.lww.com/TA/D954>).^{15,40,43} In contrast, BVI do not differ as much in their stroke risk for Grade II–IV injuries.^{15,43} In the absence of thromboembolism, a unilateral VA stenosis or occlusion may not be symptomatic if the contralateral VA provides robust flow. There are likely complex flow dynamics that are affected by VA asymmetry, atherosclerotic disease, collateral circulation, and possibly other factors. Vertebral hypoplasia is associated with posterior inferior cerebellar artery territory hypoperfusion in 42% of patients; this is usually subclinical but may be exacerbated by flow disturbance.⁸ It has been suggested that Grade I injuries are innocuous and may not warrant treatment.⁴⁴ However, the stroke risk is real. An EAST multicenter study⁴⁵ found that Grade I injuries persisting on repeat imaging had a 16% stroke risk, and Lauerma et al.⁴⁶ reported strokes in 12% of Grade I carotid injuries.

In the original grading scale, Grade II injuries encompass 25% to 99% stenosis; visible intimal flaps; or intraluminal thrombus. Lauerma et al.⁴⁶ reported an increased rate of stroke among patients with Grade II and III injuries who had >50% luminal narrowing.⁴⁶ Although the stroke difference was not statistically significant, it is compelling and consistent with our anecdotal experience. We think intraluminal thrombus and hemodynamically significant stenosis (>70%) increase the stroke risk, and propose a modified grading scale to reflect these risks within the Grade II and III injury categories (Table 2, Fig. 3). This should be validated in large numbers of patients with BCVI.

TREATMENT

Treatment is aimed at preventing stroke and promoting vessel wall healing. Strategies include antithrombotic therapy (AT), endovascular intervention (EI), and surgical repair. To

TABLE 1. Distribution of Injuries and Injury Grades Between Carotid and Vertebral Arteries, Based on High-Risk (Original Denver Criteria)¹⁵ Versus Universal screening³¹

Screening Criteria	High-Risk Screening			Universal Screening		
	Carotid	Vertebral	Total	Carotid	Vertebral	Total
Injuries (n)	157 (62%)	97 (38%)	254	317 (52%)	297 (48%)	614
Grade I	89 (57%)	48 (49%)	137 (54%)	174 (55%)	133 (45%)	307 (50%)
Grade II	31 (20%)	21 (22%)	52 (20%)	92 (29%)	79 (27%)	171 (28%)
Grade III	24 (15%)	8 (8%)	32 (13%)	38 (12%)	13 (4%)	51 (8%)
Grade IV	5 (3%)	20 (21%)	25 (10%)	13 (4%)	71 (24%)	84 (14%)
Grade V	8 (5%)	0 (0%)	8 (3%)	0 (0%)	1 (0.3%)	1 (0.2%)

date, there are no prospective randomized placebo-controlled trials to guide management, so treatment principles are based upon retrospective comparisons of treated versus untreated BCVI patients; strategies used in the management of nontraumatic cerebrovascular disorders (e.g., spontaneous dissection); and outcomes after penetrating injuries. The treatment strategy for a given individual depends upon symptoms, site of injury, injury grade, associated injuries, coagulation status, and local expertise.

Antithrombotic Therapy

In their seminal study, Fabian et al.¹¹ reported that BCVI patients—93% of whom had neurologic deficits at diagnosis—had improved neurologic outcomes with heparin treatment. Since the advent of screening, essentially every subsequent study has demonstrated improved stroke rates and neurologic outcomes among AT-treated versus untreated patients.^{5,15,16,47–52} Of note, the “untreated” subgroups often include patients who had stroke prior to the diagnosis of BCVI, and generally have more severe injuries and contraindications to treatment. Nevertheless, AT is recommended given the devastating potential of stroke.

The primary options for AT are heparin, antiplatelet drugs (AP), and oral anticoagulants (OACs, e.g., direct thrombin inhibitors, factor Xa inhibitors). There is a paucity of data regarding the treatment of BCVI with OACs. Given their safety and efficacy in other scenarios (e.g., venous thromboembolism; stroke prevention in atrial fibrillation), they warrant further study in BCVI.

There are no randomized trials comparing heparin with AP in the management of BCVI. Multiple retrospective studies suggest that AP may be at least as effective as heparin for stroke prevention.^{15,47,48} In a systematic review, stroke rates were

equivalent but hemorrhage less common with AP (2% vs. 6%).⁴⁹ However, caution is required in interpreting retrospective data. Most studies do not provide details on injury grade, drug, dosing and timing. Moreover, there may be a bias to treat more severe injuries with heparin, based on earlier literature and current guidelines.^{5,11,20,52} In a survey on BCVI management, heparin/warfarin was preferred over AP for “most cases” (43% to 33%); and for the higher-risk situation of an asymptomatic patient with intraluminal thrombus, heparin/warfarin was even more heavily favored (66% to 23%).⁵³ Lauerman et al.⁴⁶ describe AP for lower-risk BCVI and heparin for the high-risk carotid injuries (Grade II with >50% stenosis, Grade III, and Grade IV). A study from Seattle further illustrates the difficulty in interpreting retrospective data. Anticoagulation was used in 77 (11%) of 677 patients, and AP in 600 (89%).⁵⁴ The stroke rate was 12% with anticoagulation and 4% with AP, but those treated with anticoagulation had more risk factors for stroke: they were more severely injured, with more prehospital/emergency department hypotension (34% vs. 20%); had higher BCI injury grades with more Grade IV injuries (8% vs. 1%); and had more microemboli on transcranial Doppler monitoring (35% vs. 19%).⁵⁵ Concerns about the efficacy of ASA were raised by Abraham et al.⁵⁶ They reported on 12 patients who had BCVI-related strokes while hospitalized—7 were untreated while 5 (42%) were receiving ASA. The authors noted that in an earlier report from their institution, nearly half of ASA-treated patients had ischemic lesions detected on MRI.⁵⁷ In a WTA multicenter study, 64 patients were on AT at the time of stroke—73% of whom were receiving AP.¹³ Based on these considerations, we continue to recommend heparin over AP for higher-risk patients.^{20,52}

Drug Dosing and Monitoring

In early experience with heparin for BCVI, bleeding complications occurred in 54% of patients treated with a loading dose (70 U/kg) followed by titration to activated partial thromboplastin time (aPTT) 60 seconds to 80 seconds.⁵ Elimination of the loading dose and titration to aPTT 40 seconds to 50 seconds reduced the bleeding complication rate to 4%.^{11,15} Bleeding complications are currently reported in about 6% of patients receiving heparin or other anticoagulants for BCVI.⁴⁹

If heparin is contraindicated or unavailable, we recommend AP—aspirin (ASA) 325 mg or clopidogrel 75 mg daily. The therapeutic effect of AP is difficult to monitor. Nonresponsiveness is common to both ASA (24% overall)⁵⁸ and clopidogrel (21%),⁵⁹ with rates as high as 60% to 65% in

TABLE 2. Blunt Cerebrovascular Injury Modified Grading Scale

Grade	Description
I	Intimal irregularity or dissection with <25% luminal stenosis
IIa	Dissection or intramural hematoma with 25–70% luminal stenosis
IIb	Dissection or intramural hematoma with intraluminal thrombus or >70% luminal stenosis
IIIa	Pseudoaneurysm <1 cm diameter with <70% luminal stenosis, or hemodynamically insignificant arteriovenous fistula
IIIb	Pseudoaneurysm ≥1 cm diameter or with ≥70% luminal stenosis
IV	Complete occlusion
V	Transection with active extravasation or hemodynamically significant arteriovenous fistula

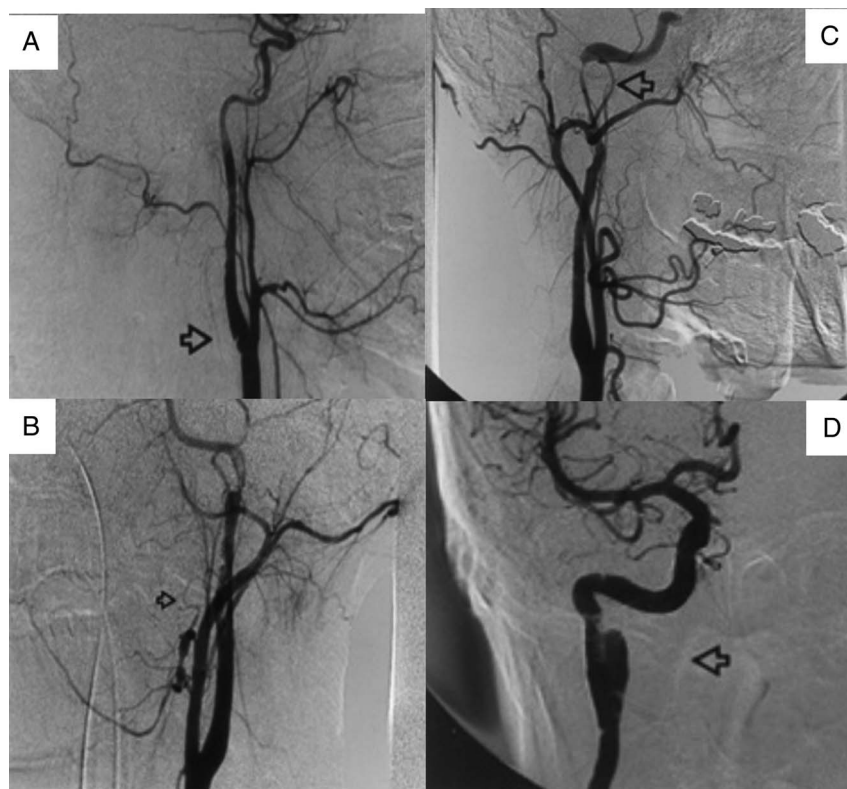


Figure 3. Grade II and III injuries. (A) Grade IIa injury; visible intimal flap (arrow). (B) Grade IIb injury; intraluminal thrombus (arrow). (C) Grade IIb injury; dissection with severe stenosis (>90%). (D) Grade IIIb injury; pseudoaneurysm >1 cm.

individual studies.⁶⁰ We favor ASA 325 mg over 81 mg as nonresponsiveness varies by dose: 36% with doses <101 mg versus 19% with >299 mg.⁵⁸ Antiplatelet resistance is a risk factor for major adverse cardiac events in patients being treated for cardiovascular disease, and ischemic events in peripheral arterial and cerebrovascular disease.⁶⁰ The relationship between AP resistance and BCVI-related strokes is unknown—but logically, the finding of AP nonresponsiveness would suggest using an alternative AT regimen. It must be emphasized that current platelet function assays have poor prognostic value for adverse events and further research is required, including the utility of viscoelastic assays.^{60,61} While dual AP may be beneficial for other conditions, we do not recommend it in BCVI due to lack of established benefit and a possible increased risk of bleeding.⁶²

Contraindications

Patients with BCVI often have associated injuries (e.g., TBI, spinal injury, solid organ injury) that contraindicate AT. A systematic review of patients with significant concomitant injuries found that AT can be safe, with hemorrhagic complications in just 3.4% of treated patients.⁵¹ The importance of treatment is highlighted by the finding of strokes in 34% of untreated versus 8% of treated patients. All contraindications are relative, and the status of the injuries is dynamic. The risk:benefit of treatment should be reassessed at least daily, as most strokes occur within 72 hours of injury.¹³ Strategies that may inform decision-making include repeat vascular imaging at an early interval to evaluate progression⁵²; MRI to look for new ischemic lesions⁵⁷;

transcranial Doppler to assess microemboli burden⁵⁵; and assessment of the coagulation profile to determine hypercoagulability.⁶³

Endovascular Interventions

The role for endovascular interventions (EI) in BCVI is evolving, as advances in technology and technical expertise offer opportunities to improve the outcomes of patients, and the definition of an “accessible lesion” has changed dramatically.^{42,64–66}

Therapeutic Interventions

Control of active hemorrhage or symptomatic arteriovenous fistulae are well-accepted indications for EI.^{40,64–66} Hemorrhage control may be achieved by endovascular stent placement or embolization with coils, liquid embolic agents, or vascular plugs.⁶⁶ In the setting of stroke due to large vessel occlusion, mechanical embolectomy is generally considered the standard of care.⁶⁷ However, there are few reports of thrombectomy in the setting of BCVI. In a recent small study, four patients had successful thrombectomy after stroke, with excellent neurologic recovery.⁴² Endovascular stents may be deployed in the setting of dissections, pseudoaneurysms, or occlusions to reconstruct injured arteries following thrombectomy. Patients with ischemic neurologic symptoms may be eligible for thrombolytic therapy within the appropriate time period (<4.5 hours), but concomitant injuries may contraindicate it. In order to optimize stent and arterial patency, AT is needed. This requires careful consideration and multidisciplinary discussion for the individual patient, and further study to determine optimal approaches to those with stroke and major bleeding risk.

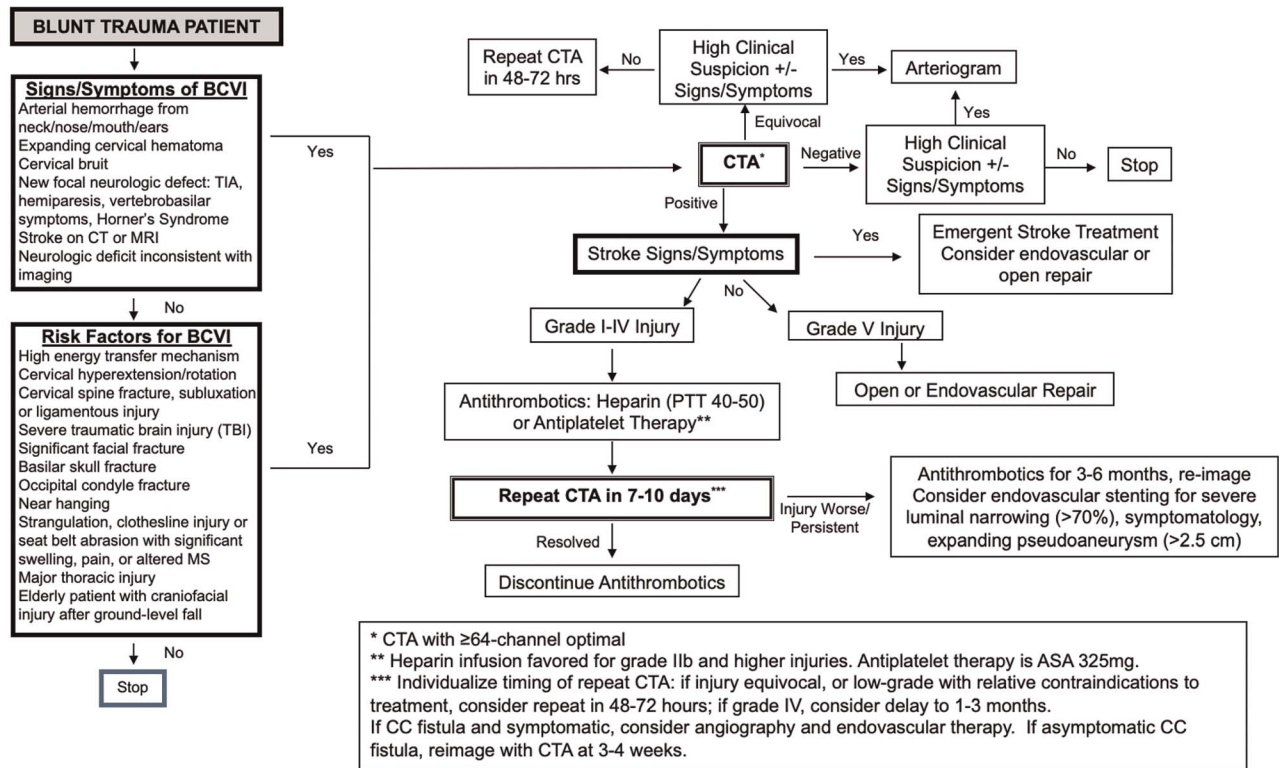


Figure 4. Algorithm for the diagnosis and management of BCVI.

Prophylactic Interventions

Most of the literature pertaining to EI in BCVI has focused on prophylactic interventions for pseudoaneurysms or significant stenoses.^{47,50,68-70} Some groups have reported a good safety profile and outcomes with endovascular stenting, but there have been no controlled trials. In the early 2000s, a report of 17% stent-related complications and 45% stent occlusion forced reconsideration of the practice.⁶⁸ Given the efficacy of AT, and the risks and expense of EI, the use of prophylactic stents has decreased significantly and is currently not recommended as routine or first-line treatment.^{21,50,69-71} We think that there is a role for selective stenting—for example, for pseudoaneurysm enlargement to >2.5 cm—but there is a paucity of data supporting specific criteria, and reports of pseudoaneurysm rupture are rare. Stent placement for significant stenosis ($>70\%$) is similarly theoretically sound but prospective data are lacking.⁷⁰ Caution is needed to minimize ischemia and avoid dislodging debris during stent deployment, and post-stent AT is mandatory. Further study is warranted to assess the true safety and efficacy of stents.

Preemptive coil embolization, primarily for vertebral arteries, was discussed over 20 years ago,^{19,64} and has been promoted in the setting of Grade IV BCVI to prevent distal embolization in the event of arterial recanalization or at the time of cervical spine surgery.^{66,72-74} A recent small series included two patients who had stroke after prophylactic VA embolization.⁴² Neither stroke was in the ipsilateral posterior circulation, but the strokes may have been a consequence of altering cerebral flow dynamics. This requires controlled study, and the risks must be considered in light of the data indicating that patients do well with AT alone.

Surgery

Recommendations for open repair of surgically-accessible high-grade injuries have been based on observational studies of patients with penetrating injuries, and blunt injuries in which patients with minimal neurologic deficits did much better with carotid artery repair compared with ligation or no treatment.^{1,2} Given the efficacy of AT, the less-accessible location of many BCVI in the VA or distal ICA, and the availability of endovascular expertise, the number of patients undergoing open surgical repair of BCVI is exceedingly small. Some high-grade injuries of the CCA or cervical ICA may require open surgical management, usually involving interposition grafting. Extracranial-intracranial bypass has been successfully employed in selected patients, but the use of this procedure for this indication is controversial.

FOLLOW-UP IMAGING AND LONG-TERM TREATMENT

Routine follow-up imaging is recommended, as it frequently leads to a change in management. In an early study, 57% of Grade I and 8% of Grade II injuries healed within 7 days to 10 days.¹⁵ Given that Grade I injuries account for one-half of BCVI, and Grade II another 20% to 28%, there are a considerable number of patients who can stop treatment.^{15,31} If a patient will not be able to return for imaging, we perform it prior to discharge. In the setting of concomitant injuries that may contraindicate AT, we re-image at 48 hours to 72 hours to inform the risk/benefit discussion. Injury progression—e.g., enlarging pseudoaneurysm or worsening stenosis—may lead to a change

in treatment such as EI or surgery. Although it has been suggested that early follow-up imaging is not necessary for high-grade injuries,^{43,75} early recanalization has been reported in 55% to 60% of Grade IV injuries in two separate studies, and may be a high risk factor for stroke.^{42,72}

We perform CTA for follow-up imaging, to allow direct comparison with the initial study. In some cases, it may be preferable to perform duplex ultrasonography to avoid contrast or radiation; MRA if simultaneous brain or spine imaging is desired; or DSA if further arterial definition is required (e.g., for carotid-cavernous fistulae) or EI is planned. Later follow-up CT scanning has been recommended at a 3-month interval; Wu et al. reported that 11% of injuries resolved between 30 days and 90 days.^{20,43,52} Some authors suggest that 90-day imaging is unnecessary as lesions are likely to remain stable. In that scenario, it is recommended to continue AT empirically.

There are no robust data to guide the choice or duration of long-term AT. Our current practice is to prescribe AP (ASA 325 mg) at discharge, given its safety profile and low cost—but outcomes data are difficult to interpret. One analysis of the Nationwide Readmission Database reported a higher rate of readmission for stroke in BCVI patients treated with AP (5.7%) versus other anticoagulants (1.8%).⁷⁶ In contrast, another study based on the same database found that AP are associated with a lower readmission rate overall, but no difference in readmission with stroke.⁷⁷ A major challenge in interpreting these two studies is the lack of detail on the specific medications, dosing, and timing. This area requires further study, including an assessment of OACs, for long-term management.

OUTCOMES

Stroke and Mortality

Stroke rates from the era prior to screening were substantially higher than contemporary series, as most injuries were diagnosed based on signs or symptoms of vascular injury or ischemic neurologic insult. Since the adoption of screening protocols and aggressive AT, the stroke rate at many centers has decreased to 5% and lower, varying based on injury grade.^{49,54,78} Unfortunately, while the stroke rates are lower, the stroke outcomes remain devastating. The earliest large studies of BCVI-related strokes reported mortality of 23% to 28%, with 48% to 58% of survivors suffering permanent severe neurologic deficits.^{1–4,11} These numbers have not improved over time—contemporary multicenter studies report 32% to 40% mortality after stroke.^{13,50}

Fabian et al.¹¹ reported improved neurologic outcomes after symptomatic BCI with heparin therapy. In the WTA multicenter trial of BCVI stroke patients, 54% had neurologic improvement.¹³ Those receiving systemic heparin had improvement in 68% of cases; of those receiving AP without heparin, 52% had improvement. The optimal treatment regimen requires further study.

Functional Outcomes

A study of consecutive BCVI patients from 1996 to 2005 gathered clinical outcome data on 55 surviving patients at a mean 34 months after discharge.⁴⁷ Glasgow Outcome Scale scores were the same or improved from discharge in 95% of patients. A subsequent study from the same center assessed functional outcomes of BCVI survivors.⁷⁹ The function scores were affected

by TBI, and likely other associated injuries; they did not differ between BCI and BVI. Patients who had stroke had worse outcomes. There is no evidence that BCVI, in the absence of stroke, are independently associated with adverse functional outcomes.

SUMMARY AND RECOMMENDATIONS

Blunt cerebrovascular injuries have the potential for disastrous outcomes. They may occur following any trauma, with occurrence increasing with higher energy transfer. If stroke occurs, prompt recognition and treatment offers the best chance for optimal outcome.

Early diagnosis and provision of AT may prevent strokes, and we recommend screening of asymptomatic patients. Each institution should establish formal screening guidelines that are aligned with published recommendations. At a minimum they should involve high-risk criteria but should also acknowledge that BCVI will be missed, and expand the criteria based on injury mechanism.

Early provision of AT is recommended. There may be equivalence between AP and anticoagulation, but providers should recognize that heparin has historically been used in higher-risk patients, and interpret the data accordingly. We recommend heparin in high-risk patients. In patients with AT contraindications, consider early reimaging, MRI, transcranial Doppler, or hypercoagulability assessments to inform decision-making.

Multidisciplinary communication and decision-making are critical in navigating decision-making, particularly related to contraindications and potential EI. Figure 4 is a recommended algorithm for diagnosis and management.

AUTHORSHIP

W.L.B., E.E.M. participated in the conception and study design. W.L.B., E.E.M., A.P.K., B.C.C.R.F., J.S.W. participated in the literature review. W.L.B., E.E.M., A.P.K. participated in the drafting of the article. W.L.B., E.E.M., A.P.K., B.C.C.R.F., J.S.W. participated in the critical revision.

DISCLOSURE

Conflicts of Interest: Author Disclosure forms have been supplied and are provided as Supplemental Digital Content (<http://links.lww.com/TA/D955>).

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